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Abstract

Although the impact of neurodegenerative diseases on everyday life interactions is well known in the literature, their impact on social cognition processes remains unclear. The concept of social cognition refers to a set of skills, all of which are essential to live in a group. It involves social knowledge, perception and processing of social cues, and the representation of mental states. This article reviews recent findings on the impact of cortical and subcortical neurodegenerative diseases on three social cognitive processes, namely theory of mind, empathy and emotional processing. We focus on a conceptual approach to each of these skills and their cerebral underpinning.

Keywords	social cognition; neurodegenerative diseases; theory of mind; empathy; emotion recognition; neuropsychology.
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Theory of mind, empathy and emotion perception in cortical and subcortical neurodegenerative diseases

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Abstract. Although the impact of neurodegenerative diseases on everyday life interactions is well known in the literature, their impact on social cognition processes remains unclear. The concept of social cognition refers to a set of skills, all of which are essential to live in a group. It involves social knowledge, perception and processing of social cues, and the representation of mental states. This article reviews recent findings on the impact of cortical and subcortical neurodegenerative diseases on three social cognitive processes, namely theory of mind, empathy and emotional processing. We focus on a conceptual approach to each of these skills and their cerebral underpinning.

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Social cognition: definition

Human relationships require adaptation to others by the intervention of socio-cognitive and emotional processes to regulate the behavior in the interaction. These processes represent the field of research of social cognition, which is of growing interest in the scientific literature. In recent decades, studies in social neuroscience have particularly developed, especially after the 2000s (Figure 1).

This enthusiasm is not surprising considering that social relationships are at the heart of human activity and that some behavioral disorders demonstrated by brain-damaged patients are likely to be explained by an impairment of social cognition processes.

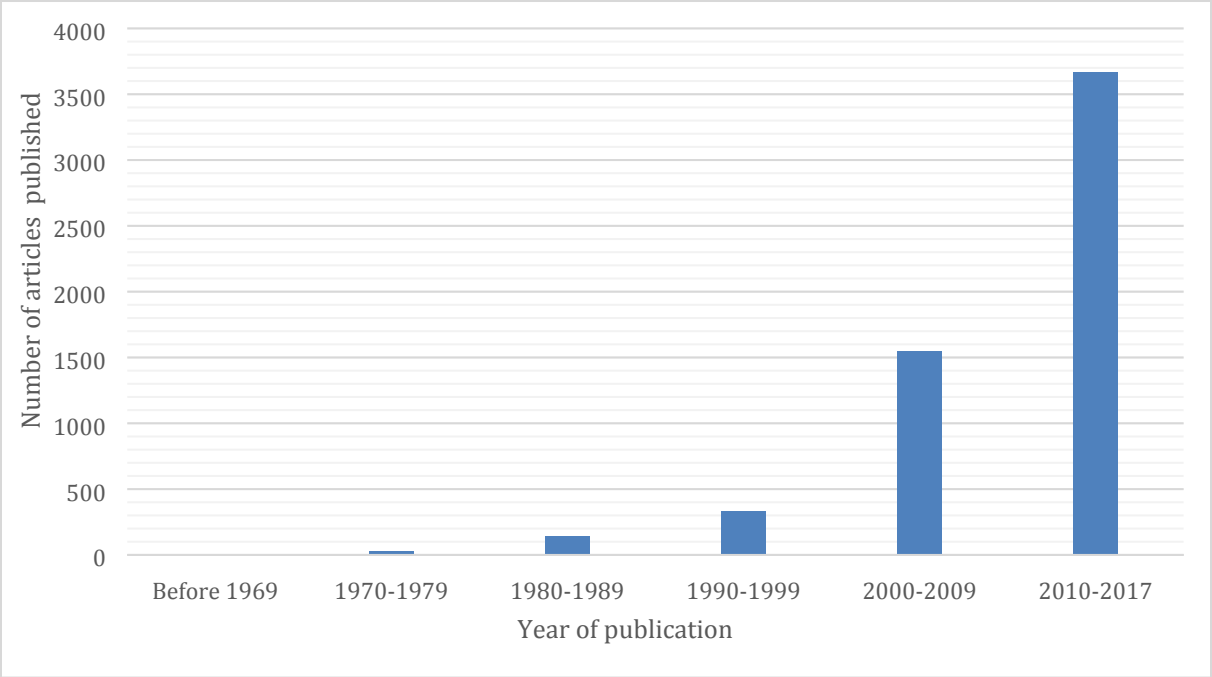

Figure 1: Number of articles in *Pubmed* containing "social cognition" in their titles between 1967 and 2017.

Most of this research has focused on the definition of the cognitive architecture of social cognition processes, their dissociation and their relationship with other components (e.g. executive functions). Most results tend to show that a deficit in social cognition always leads to a behavioral disorder in neurological [1] or psychiatric diseases [2].

Social cognition includes all the socio-emotional capacities that enable the monitoring of behavior in an interaction situation. In our view, Frith's [3] definition of social cognition brings together most of these key points: "*Social cognition concerns the various psychological processes that enable individuals to take advantage of being part of a social group. Of major importance to social cognition are the various social signals that enable us to learn about the world."* (Frith, 2008, p. 2033).

According to this definition, social cognition is composed of several different concepts. Theory of mind, empathy and emotional regulation seem to be the most studied in the literature.

Theory of mind

There is no consensus about the definition of Theory of Mind (TOM) in the literature. Some authors see it as the ability to access the mental states of others, whereas others define it as the ability to adopt the point of view of others [4]. In neuropsychology, although the term TOM refers to several meanings, it is generally considered to represent the meta-cognitive abilities that facilitate the implementation of social interactions. These meta-representational capacities are enabled by an inference system leading to the attribution of mental states such as thoughts, intentions, beliefs or feelings.

Several dissociations of the components of TOM are available. A major one refers to automatic and controlled processes and echoes the proposals of Sabbagh et al. [5] and Apperly and Butterfill [6]. Automatic processes are considered as rapid and not necessarily requiring language, in contrast to controlled processes, which are slower and effortful [7,8]. In this review, we focus only on controlled processes.

Another dissociation was defined by Coricelli [9] who distinguished cognitive TOM, i.e. making inferences about the epistemic mental states of others without emotional involvement, from emotional TOM, which defines empathy as the cognitive ability needed to understand the emotional feelings of others (see below).

Concerning the neuroanatomical substrates thought to underlie the TOM process, they have been the subject of numerous works and are still being debated (see [10,11] for a review), but it seems possible to identify the brain structures frequently mentioned in these

studies, classified into three groups. The first refers to the limbic and para-limbic regions, which include the orbitofrontal cortex, the amygdala and the anterior cingulate gyrus. These regions underpin the skills associated with the emotional side of TOM [12]. Stone et al. [13] confirmed these results, showing that patients affected in the frontal-orbital regions were less efficient than patients with dorsolateral frontal lesions in a "*faux pas*" task [13] requiring emotional responses. However, these patients were able to verbalize the emotional states or beliefs of the protagonists, unlike patients with dorsolateral lesions. Thus, this meta-representational aspect, comparable to the cognitive side of TOM, seems to depend more on dorsolateral prefrontal structures [9]. The second group involves the medial prefrontal regions. Gallagher et al. [14] reported the activation of the medial frontal gyrus only when processing designs requires the mobilization of cognitive TOM abilities (answers regarding the beliefs or intentions of protagonists) compared to neutral designs (see also [15]). The third group involves the posterior brain regions that include the right posterior parietal system and, in particular, the inferior parietal lobule and the right superior temporal sulcus and the temporal poles. Although there is a consensus about most of these neuroanatomical substrates, there are very contrasting views in the literature about one area. Many authors agree that the temporoparietal junction (TPJ) is involved in TOM, but there is debate about the lateralization. Based on case studies or meta-analysis, authors cannot agree whether the right TPJ [16–18] or the left TPJ [19–21] is essential for TOM. There is clearly a need for further studies on this topic.

In short, functional imaging data tend to confirm the dissociation between cognitive and affective TOM: the cognitive side of TOM is supported by the dorsomedial and dorsolateral prefrontal cortex while the emotional side is supported by the ventromedial and orbital frontal regions. [22]. More recently, it has been proposed that the dorsal striatum, the dorsal temporal pole and the dorsal anterior cingulate cortex underlie the cognitive TOM

network, whereas the ventral striatum, amygdala, ventral temporal pole and the ventral anterior cingulate cortex underpin the affective TOM network [23].

Empathy

Empathy specifically refers to the process of developing inferences about the emotional states of others, to share and/or feel their emotions. Decety and Lamm [24] defined empathy as the ability to understand what others think, but also to experience it, without confusion with oneself. Like TOM, empathy is a process involving several components, which has led some authors to distinguish emotional (also referred to as emotional contagion) and cognitive empathy [25,26]. Cognitive empathy is defined as the ability to take the perspective of others and understand what he/she feels but without experiencing it, while affective empathy refers to the ability to feel the emotional states of others. The emotional component (low-level process) is directly associated with emotional resonance, a term used to define the fact that the observation of an emotion in another person is likely to induce the same emotion for the observer by activating identical representation. The cognitive component of empathy involves high-level processes such as executive functions (especially inhibition), as well as the ability to distinguish oneself from others, to adopt the other's perspective and assign thoughts and emotions [24]. The terms affective TOM and cognitive empathy therefore seem to refer to similar concepts [27].

Concerning the neuroanatomical correlates of empathy, the emotional component is mainly associated with the mirror neuron system [28], while the cognitive component involves the inferior parietal lobule [25]. Some authors have also noted that the neuronal activations of cognitive empathy are identical to those of affective TOM and that the brain regions recruited during a test with emotional TOM inference are identical to those used

during empathy tests [27], thus corroborating the idea that cognitive empathy and affective TOM are two overlapping concepts.

Emotional processing and emotional regulation

Because it has a social and a cultural dimension, emotion is a signal that can be used to modify the actions of the partner, as well as of the individual him/herself, which was first explained by Darwin [29]. The activation of the mirror neuron system when someone sees the emotion of another person can induce the same emotion for the observer [30].

Humans can detect both "basic" facial expressions and those reflecting complex mental states that regulate social interactions (which seem hostile or friendly) or indicate the thoughts of others (which seem pensive). Ekman's work [31] identified seven universal emotions, independent of cultural aspects (anger, disgust, joy, neutrality, fear, surprise and sadness). Hornak et al. [32] showed the presence of disturbances in the identification of facial and vocal emotional expressions in a group of patients who had inadequate social behavior. An alteration in the identification of emotional expressions was correlated with the degree of change in the subjective emotional feeling evaluated by patients. Moreover, some studies have reported a strong positive correlation between this feeling and behavioral problems. Thus, the inability to decode emotional expressions seems to harm behavioral control (social adjustment, euphoria, irritability) given the fact that emotion then no longer acts as a censor to adjust environmental behavior.

Social cognition in pathological aging

Social cognition disorders lead to patients becoming isolated from their relatives as well as from the medical staff [4]. A review of the various neurological pathologies that affect social cognition therefore seems important in order to study the potential specificity of the

deficits according to the pathology. In this section, we report the evolution of the main components of social cognition (TOM, empathy and emotion perception) in various cortical and subcortical neurodegenerative diseases. In our view, we can hypothesize that distinguishing the diseases as a function of their etiologies is interesting due to the fact that it is well known that cortical and subcortical diseases lead to several patterns of disorders [33].

Cortical diseases

Frontotemporal dementias

The behavioral variant of frontotemporal dementia (bvFTD) is the most frequent form of frontotemporal dementias (FTDs) [34]. It is characterized by an insidious beginning with a gradual evolution reaching first the frontal lobes, causing a change in social and interpersonal behaviors. The loss of gray matter mainly occurs in the frontal lobes, specifically in the orbital regions (Brodmann area (BA) 10, 11, 47), and the medial and dorsolateral regions (BA 8, 9, 46), followed by a loss in the temporal lobes [35]. It is also characterized by various changes in executive functions and a lack of awareness of deficits (anosognosia) [36,37].

As mentioned above, changes in interpersonal behavior can be explained by an impairment of social cognition abilities. This has been the subject of a growing number of publications in recent years, [38,39] including TOM abilities. To assess these, authors have used a wide range of tasks, including *faux pas,* cartoon tasks and first and second order false belief reasoning tasks. The first order refers to the inference made by a person about the mental or belief state of a second person in a story, while the second order refers to the participant's inference about the mental state or belief of a first person concerning the inference or belief of a third person. Results tend to show a major deficit in first and second order TOM capabilities [40,41] in patients with bvFTD, which may explain, at least in part, changes in the relationships of patients. For some authors, this TOM deficit is not due to a general cognitive deficit [38,42],

but rather to a deficit in inhibiting their own perspective [38], according to the Samson model [20] explained earlier.

Another way to explain the behavioral problems of bvFTD patients could be a cognitive and/or affective empathy deficit, which could prevent patients from correctly feeling the emotions of others.

The Interpersonal Reactivity Index (IRI) questionnaire [43,44] seems to be one of the most commonly used in the literature to assess empathy [41]. The results of research in this field are consistent and show a decline in these abilities on both the cognitive and the emotional side [40]. Empathic disorders are considered a diagnostic criterion for this disease and could lead to a change in the lives of patients for the decisions and judgments they make. From an anatomical point of view, these disorders can be explained by a reduction in gray matter in the frontal areas, amygdala region, left insula and the posterior-superior temporal sulcus at the TPJ [45,46].

Recognition of emotions also appears to be impaired in bvFTD. Using classic recognition tasks, such as Ekman's [31], most studies confirm a deficit for all basic emotions [47,48] for these patients compared to control subjects.

To summarize, it seems that all social cognition processes are affected in bvFTD. This set of problems leads to the inability of patients to communicate with their families, for which the management of social cognition disorders seems essential. It has been shown that, for people suffering from a traumatic brain injury [49] or from neurological diseases such as schizophrenia [2,50], rehabilitation therapies can have a positive impact on their interactive skills and psychosocial abilities. It therefore seems likely that this kind of rehabilitation could be suitable for bvFTD patients to try to, at least, stabilize their psychosocial skills.

Alzheimer's disease

Alzheimer's disease (AD) is the most common form of dementia. According to the *Alzheimer's Disease International Group*, in 2015 there were nearly 44 million people living with Alzheimer's disease or related dementia worldwide. AD is characterized mainly by an accumulation of amyloid beta proteins, causing the emergence of amyloid plaques, and by neurofibrillary degeneration due to the Tau protein. These neuronal damages mainly cause hippocampal atrophy in the early stages of the disease, and then spread to the entire cerebral cortex. In the literature, many studies report quite controversial results about a potential TOM deficit in AD.

Many authors [38,51,52] conclude that there is a TOM deficit in AD, but confined to the second order task for false beliefs, while others claim that a memory deficit is responsible, especially in working memory in such tasks as they involve a high cognitive load [52]. Moreover, according to Samson's [19,20] model, which locates the inference of the perspective of others in the TPJ area, which is damaged early in AD, a deficit in a false belief task could be explained by a specific impairment of this capacity. A recent data-mining study [53] examined the potential link between scores on executive function (EF) tasks and those on TOM tasks in patients with AD. The authors found that around 49% of TOM performance can be predicted by EF measures. In other words, according to these authors, it seems that the decline in TOM observed by some studies may not be a specific domain decline but rather a more general, mainly executive, decline. However, in contrast, some authors [38,42,54] have found that despite the impact on EF, AD patients present a *pure* TOM deficit. More precisely, Le Bouc et al. [38] found hypometabolism in the left TPJ, supporting Samson's [19,20] point of view that the TPJ is essential for belief inference. It therefore seems that a TPJ deficit is a key point in understanding TOM disorders in patients.

Regarding cognitive and emotional empathy abilities, the results are still very divergent in AD. Some authors report a preservation of the capacity for empathy in these patients [55] while others obtain opposite results [44,56]. This discrepancy could be explained by the inclusion of patients at different stages of severity.

Studies on emotion detection also show some contradictions. Some report a deficit in emotion detection on faces [57,58] while others describe the preservation of this capacity [59,60]. However, Burnham's study [59] provides a nuance. In a perception of emotions task, AD patients successfully combined the emotion of a face with the corresponding emotion written in a list, but were unable to connect two photos with the same emotion. The authors therefore concluded that AD patients could potentially have a spared perception of emotions but may present visuo-spatial disorders. Furthermore, a recent meta-analysis [61] considered that, even when cognitive status is controlled, AD patients always seem impaired in their emotion decoding abilities.

It is thus difficult to conclude that there is a deficit or a preservation of social cognition skills in AD. Regarding TOM, the first order tasks are mostly successful in AD patients (but see [54]), which means that a part of TOM is spared by the disease. For other components of social cognition, it is difficult to draw a conclusion, given that both the tasks and the recruitment of patients differ between studies.

Amyotrophic lateral sclerosis (ALS)

Amyotrophic lateral sclerosis (ALS), sometimes known as *Charcot's disease*, is a degenerative disease of unknown origin that mainly affects the motor abilities of patients. It primarily destroys motor neurons in both the central and peripheral systems [62,63]. In about 30% of cases it begins in the brain stem, hindering patients from speaking or swallowing, whereas for the other 70% it starts with a spinal form, hampering the voluntary movements of

the limbs [64]. In all cases, patients end up with general palsy, including of the respiratory muscles, obliging them to have heavy equipment. This disease should be differentiated from Charcot-Marie-Tooth disease, a genetic disorder that also affects motor function. A Scottish study [65] showed that 35% of patients appear to suffer from language disorders, and 23% from fluency as well as executive function disorders. In addition, when caregivers were also questioned, 40% described patients as undergoing behavioral changes. Despite these deficits making the evaluation more complex, research studies began a few years ago [63] and have hugely increased in recent years [66,67] (see [68] for a review).

Concerning the theory of mind, studies using the RME [69] or the faux pas [13] test found a significant difference between dysexecutive patients with ALS and control subjects [66,67,70]. However, these results have led to debate. Some authors report that this difference cannot be found when ALS patients without executive disorder are compared with control subjects [66,67,71], whereas others find that this difference persists when executive scores are entered as covariate [71] (see [68] for a review). These results are highly interesting for the debate about the relationship between executive functions and TOM [72,73].

Regarding the empathic capacity of ALS patients, studies are relatively rare, or even absent from a recent meta-analysis on social cognition and ALS [68]. To our knowledge, only one study has examined it [71]. In this research, ALS patients (20% with executive dysfunction) had to attribute emotions to comic strips. The results showed a significantly lower score for ALS patients compared to control subjects. Facial recognition with Ekman's task [31] seemed to be affected too in ALS patients compared to controls [74,75] (see [68] for a review). Concerning prosody emotion recognition, the studies are both rare and heterogeneous with one reporting a deficit [76] and one nothing [74].

To sum up, it seems difficult to draw a conclusion about the possible impact of ALS on social cognition skills. For TOM, although it appears to be affected, there is still debate about the impact of the executive dysfunction. Concerning the other components, the lack of research results prevents a conclusion.

Subcortical diseases

Parkinson's disease

Parkinson's disease (PD) is caused by the degeneration of dopaminergic neurons in the substantia nigra, striatum, thalamus and subthalamic nucleus, which leads to frontal-striatal loops [77]. From a neuropsychological point of view, a large variety of disorders is observed. However, it seems that the majority of patients show executive disorders [78] and that at least 80% of patients suffer from dementia late in the disease progression [79]. Beyond motor disorders that massively hamper patients' lives, some studies have shown behavioral and psycho-emotional changes, such as depression, apathy or anxiety [80].

Studies on TOM disorders agree that there is a deficit in TOM in non-demented PD patients [72,81,82]. Like for TOM, there is a consensus in the literature about impaired empathy skills among PD patients in the early stages of the disease [83]. From an anatomical point of view, the deterioration of TOM and empathy in PD patients seems inevitable due to the damage of frontal striatal loops and amygdala [84].

Most studies on the recognition of emotions report a deficit in PD, but not all agree about an overall impairment. In many studies, not all emotions seem to be recognized. For example, Assogna et al. [85] reported a circumscribed deficit in the recognition of disgust and a preservation for other emotions, while Lawrence et al. [86] found only a lack in the recognition of anger. A meta-analysis of emotion recognition in PD [87] concluded that only a

certain proportion of patients seems to have an emotion recognition disorder, mainly for negative emotions and especially for disgust. According to these results, in a sample of people in which half are PD patients and half are healthy then statistically 63% of patients will have an emotion recognition disorder *versus* 37% for controls. The authors warn that emotion recognition has high inter-individual variability. Regarding the recognition of emotions through language, a meta-analysis [88] concluded there was a prosody recognition deficit in PD patients.

A recent study [89] found a link between the facial emotion deficit and apathy in PD patients. Another found an overall deficit without a link to general cognitive abilities [90].

In summary, it seems that most studies find social cognition deficits in PD patients with the exception of Rosen's study [91].

Huntington's disease

Huntington's disease (HD) is a neurodegenerative pathology [92] with a genetic origin, resulting in physical and intellectual harm. Patient disorders are characterized mainly by physical impairment with the occurrence of involuntary, rapid and irregular movements (chorea) between 35 and 50 years old. Cerebral involvement of HD is characterized by a decrease in the volume of subcortical regions (the putamen and caudate nucleus), striatum and cortical regions, progressing from the temporal lobes to the frontal lobes [93].

Allain et al. [94] showed that beyond the executive disorders, frontal lobe involvement in HD also provoked social cognition disorders. In their paper, the authors focused on TOM disorders using both a non-verbal assignment of intentions and Baron-Cohen's "Reading the Mind in the Eyes" test [69]. The task assigning intentions consisted of 56 small cartoons, half requiring inference of the intentions of the characters, the other half requiring understanding of physical causal relationships. The participants were instructed to choose an image in order

to complete the sequence. To do this, they had to infer the intentions of the characters with simple and realistic first-order intentions [95]. The RME consisted of 36 photographs of the eye region of different actors. Participants had to find which of five words (hatred, panic, guilt, reflection and admiration) best described what the person in the photo was thinking about. This task requires participants to put themselves in the person's place in the photograph while assigning them complex mental states.

The results showed a significant decrease in the performance of HD participants in both trials compared to a group of elderly controls [94]. This decrease reflects a deficit in both the cognitive dimension (attribution of intentions) and the emotional dimension (reading emotions in the gaze) of TOM. Several other studies have shown that performances in TOM tasks in patients with HD are reduced (see [96] for a review). Adjeroud et al. [95] measured the performance of TOM in pre-symptomatic and symptomatic stages. The originality of their study was the use of a modified version of the French "Yoni" test [97] evaluating emotional and cognitive TOM in 1st and 2nd order, with a control condition. Symptomatic patients had difficulty in inferring mental states in 1st and 2nd order, affective and cognitive dimensions, while pre-symptomatic patients presented performances similar to controls. In addition, the authors found no correlation between TOM and executive functions. A meta-analysis [98] also showed cognitive and affective TOM deficits in symptomatic patients, the latter having more difficulty than pre-symptomatic patients. In the pre-symptomatic stage of the disease, a high probability of motor symptoms in the first 5 years is associated with a greater deficit in TOM, suggesting that performance in social cognition could be a marker of disease progression.

Empathy, assessed by the Interpersonal Reactivity Index [43] or empathy scale [99], seems preserved in HD [73], but only a few studies have assessed it. Trinkler et al. [100] also

showed that patients with HD had an intact understanding of their own feelings and were able to infer the feelings of others in an empathy test.

Impaired emotion recognition is highlighted in patients with HD at the pre-symptomatic stage [98,101]. The meta-analysis of Bora et al. [98] shows that the recognition of six basic emotions is impaired in these patients, including anger, disgust and fear. Symptomatic patients are more affected than pre-symptomatic patients. Evaluation methods of emotion recognition included photographs of faces, emotion hexagons, short videos and vocal sounds, showing that impaired emotion recognition concerns not only facial expressions but also body gestures and emotional prosody [102].

In summary, patients with HD appear to undergo alterations in the cognitive and affective dimensions of TOM as well as in the perception of emotions, whereas empathy seems to be preserved.

Multiple sclerosis (MS)

Multiple sclerosis is an autoimmune inflammatory disease affecting the myelin sheath of the central nervous system, white matter and gray matter [103]. It affects a large number of cerebral regions without following a typical pattern of progression [104], leading to a large number of cognitive deficits [105–107]. The quality of life tests show particularly low scores for MS [108], so it seems important to explain which variables are responsible for these problems. Although motor problems clearly play a preponderant role, some cognitive variables, such as social cognition, also seem to be involved [109].

Among the social cognition functions, TOM seems to present a deficit when evaluated with the RME [110], the *faux pas* test [111] or movies [112].

Regarding empathy, patients also appear to have a significant deficit in both cognitive and emotional empathy [87, 88]. However, some authors [111] have shown that at the onset of the disease, patients describe themselves as more empathic than the average. This deficit could be explained by anosognosia, a deficit that appears to be present in a large proportion of patients [114]. In relation to empathy, the majority of studies in patients with MS appear to show a deficit in emotion recognition [107,113,114]. However, it should be pointed out that, according to some authors, this lack of emotional recognition is not generalized to all emotions, but rather to anger and fear [110].

In conclusion, it seems that all three main components of social cognition are impaired in MS. In addition to the motor syndrome, this deficit could explain the poor quality of life reported by patients [108].

Conclusion

The aim of this article was to present the definitions of the most studied social cognition processes, their main cerebral foundations and, above all, the effects of cortical and subcortical neurodegenerative diseases on the main social cognition skills.

Our literature review shows that there is sometimes a close relationship between the different processes of social cognition, both for their definitions that may overlap and for the cerebral structures that underlie them. This work also provides a better understanding of the links between social cognitive skills and other cognitive skills, mainly including executive functions. Nevertheless, several points need to be raised. Firstly, while data relating to social cognition seem important for some pathologies, mainly schizophrenia and autism [2,115–117], there is significantly less research for other disorders, particularly neurodegenerative

diseases. Secondly, while many authors associate social cognition with the ability to behave appropriately in a situation of interaction, few studies have validated this proposition.

According to the different studies, it seems that social cognition processes are affected, at least in part, in cortical and subcortical neurodegenerative diseases and that in every disease mentioned, at least one process is affected. So, contrary to our expectation, there is no difference between a cortical and a subcortical etiology in terms of social cognition deficit. This absence of difference could be explained by the extent of the pathology at the time of the examination. The comparison between different diseases on objective measures being often complex. For example, the studies of Ramanan et al., (2016) [73] and Gregory et al., (2002) [52], studying TOM with FTD patients, seem to present some dissonance in the population. The first one have patients aged in average of 65.25 years old compared to 58.6 years old for the second one. The most important difference is that of MMSE passing from 24.47 to 26.66. It's possible to imagine that this difference could account to explain the relative heterogeneity between studies. This difference, beyond a change in the neuropsychological examination, could also have an impact on the brain.

For example, although AD and PD have two different etiologies (cortical and subcortical, respectively), in the end they will both reach the frontal lobe, often defined as a major lobe for social cognition. So, this etiological dissociation does not seem to be the most appropriate to analyze social cognition disorders. A classification based on the locus of a pathology does not seem appropriate either, since social cognition appears to have a wide range of neuronal bases. It may be that focusing on problems of everyday life functioning would be more relevant to distinguish patients. Moreover, the links between social cognition skills and personality or behavioral disorders remain to be explored in neurodegenerative diseases to understand whether the disturbances in social cognition processes could account for behavioral regulation deficits in interaction situations.

One way of examining the relationships between socio-cognitive disturbances and behavioral disorders in social interactions is through questionnaires. An alternative would be to think of new methodologies for evaluating social cognition that could assess the ability of subjects to analyze real interaction situations. However, most studies of social cognition processes involve classic tests or functional imaging paradigms. These are non-ecological situations, which, by definition, limit the possibilities of actually assessing the processes at work in social interactions. The methods put forward by Stuss et al. [118] or Freedman et al. [42] that involve taking a visual perspective or the detection of deception could, from this point of view, constitute a more original approach. A methodology combining social psychology and neuropsychology could also be more relevant for the emergence of a genuine social neuropsychology. An alternative could be the use of the interactionist approach [119–121], which considers that, to test social cognition processes, people need to interact with other people. In contrast to the other tests, which are often not ecological, this approach aims to assess processes as close as possible to everyday life situations.

Disclosure of interest:

The authors declare that they have no competing interest.

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